

States of Enhanced Activity in a Network of Pulse Coupled Oscillators with Dynamic Coupling

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We investigate states of enhanced activity in a biological neuronal network composed of pulse-coupled oscillators. The synaptic couplings between the neurons are dynamic, modeling spike time dependent plasticity. The network exhibits statistical characteristics which recently have been identified in an analysis of epileptic seizures [Osorio et al., Phys. Rev. E **82**, 021919(2010)] based on analogies to the onset of earth quakes.

I. INTRODUCTION

The origin of epileptic seizures is still a mystery (see e.g. [1]). The epileptic brain exhibits spatio-temporal complexity, both during seizures as well as in the inter-seizure intervals. In recent years, the dynamical properties of inter-seizure states have been in the focus of research with the aim to formulate suitable measures for the prediction of seizure onset [2].

A major goal of theoretical seizure modeling is the development of models of neuronal activity, which are able to reproduce the observations and allow for a theoretical interpretation of the spatio-temporal behaviour of seizure states. A successful model will shed considerable light on the questions whether seizures are predictable and what measures can be rewarding. The basic issues of theoretical seizure modeling are to investigate how epileptic seizures emerge, spread, and terminate in such models. Dynamical systems theory points to the importance of synchronization, however, it seems to be rather unclear whether the synchronization of neuronal activity is a by-product, whether it induces or whether it terminates a seizure [3, 4].

Recently, Osorio et al. [5] opened up a new perspective for our understanding of epilepsy assessing statistical similarities between epileptic seizures and the onset of earthquakes. Based on a comparative study of seizure and earth quake data, the authors showed ample evidence that the Gutenberg-Richter law, the Omori law and the statistics of interevent times formulated for earth quake data can be detected in epileptic seizure data as well.

The purpose of the present article is to develop a neural network model, which is able to reproduce the main characteristics of the analysis of Osorio et al. [5]. The developed model is based on a generalization of Haken's Lighthouse model (for a review we refer the reader to [6]), which is augmented by a suitable learning algorithm taking into account the phenomenon of spike-timing-dependent plasticity (STDP), following closely the work of Chen et al. [7]. The model presented in this article can be viewed as a model for epileptic seizures similar to the Burridge-Knoppoff model [8] and its variants for earth quakes. Our extension of the Lighthouse model allows for the inclusion of a spontaneous restructuring of the network architecture, making use of the idea that dynamical seizures of the brain can be learned or dislearned [9], [10].

It is well-known that adaptive networks may lead to the emergence of critical states in neural network models based on the phenomenon of self-organized criticality (SOC), as described by P. Bak [11]. Experimentally, Beggs and Plenz [12] showed convincing evidence that the dynamics of neuronal populations could actually exhibit critical dynamics by an explicit investigation of events called neuronal avalanches. Neuronal avalanches can be found in regions of control parameter space located in between a regime of coherent, wavelike neuronal activity and a regime, where neurons behave asynchronous

and spike incoherently. The existence of SOC in an integrate and fire model including dynamical synapses has been shown by A. Levina et al. [13, 14]. The dynamics of their model exhibits the occurrence of critical avalanches and the distribution of the size of the occurred avalanches showed a power law behaviour. In the case of static synapses, the coupling strengths have to be fine-tuned in order to generate a critical state, whereas dynamical synapses renders the system critical. Similar results were obtained by L. de Arcangelis et al. [15]. C. Meisel et al. [16] presented a study of neural networks including spike-time-dependent synaptic plasticity, extending the earlier work of Bornholdt and Rohlf [17]. They showed that due to the inclusion of this learning mechanism the network self-organizes into a critical state. Millman et al. [18] demonstrated the existence of SOC in nonconservative models of networks of leaky integrate and fire neurons with short-term synaptic depression. They demonstrated the existence of two states, up and down states and demonstrated that up states are critical, whereas down states are subcritical.

With respect to epileptic seizures it is not clear whether SOC plays a dominant role. Recently, it has been discussed that adaptive self-organized criticality fails during epileptic seizure attacks [19]

The present article is outlined as follows. In the first section, we review Haken's Lighthouse model and discuss its extension taking into account a learning mechanism based on the mechanism of spike-timing-dependent plasticity. In the second section, we investigate in detail states of enhanced activity. We reproduce features similar to the ones presented in [14, 20]. Then, we focus on similarities with the investigations of experimental recordings of seizure states discussed in [5]. A central issue is the question why states of enhanced activity emerge and terminate in the used model.

II. MODEL AND IMPLEMENTATION

A. Lighthouse Model

The Lighthouse model has been introduced by Herman Haken and is described in great detail in his monograph [6]. It models brain tissue as a neuronal network composed of pulse coupled oscillators. Each neuronal oscillator, labeled by the index m , is characterized by its phase ϕ_m , a 2π -periodic variable, and a dendritic current ψ_m .

The phase describes the action potential of a neuron. If the phase reaches the threshold 2π , a neuron will spike. The action potential S_k generated by neuron k is obtained as a sum over delta functions:

$$S_k(\phi_k(t)) = \sum_n \delta(2\pi - \phi_k(t_n)) \dot{\phi}_k(t) \quad (1)$$

The temporal evolution of the phase is determined by the equation

$$\dot{\phi}_m(t) = \Xi \left(\sum_k c_{mk} \psi_k(t) + p_{(ext,m)}(t), \Theta \right) \quad (2)$$

where $\Xi(\sum_k c_{mk} \psi_k(t) + p_{(ext,m)}(t), \Theta)$ is a sigmoidal function of the sum of all dendritic currents ψ_k of neurons k multiplied by a weighting factor c_{mk} and an external signal $p_{(ext,m)}(t)$. The factors c_{mk} form the increment matrix \bar{c} . In the following, we use a diagonal increment matrix $c_{mk} = c_m \delta_{mk}$. The sigmoidal function $\Xi(X, \Theta)$ is taken as the Naka-Rushton relation and describes the response of a neuron to an input current.

$$\Xi(X, \Theta) = \frac{\nu X^M}{\Theta^M + X^M} \quad (3)$$

For high input values X the output saturates at a maximal firing rate ν . If the input signal is below a certain threshold value Θ , the output will tend to zero. As outlined in [6] this models the all-or-none-behaviour of the axon hill, which generates the action potential. Due to the saturation of the Naka-Rushton relation for large input values, a refractory period of the neurons is included in the system. The constant M is related to the slope of the function. In our treatment we have used $M = 3$ and a maximal firing rate ν of 1.

When a neuron generates an action potential, this will be transferred to all connected neurons. The dendritic current $\psi_m(t)$ of neuron m will change due to the input from neuron k . It obeys the following differential equation

$$\dot{\psi}_m(t) = \sum_k a_{mk} S_k(\phi_k(t)) - \gamma \psi_m(t). \quad (4)$$

The synaptic weights are denoted by the matrix elements a_{mk} and describe the strength of the connection from neuron k to neuron m . The synaptic weights characterize the connections of the neural network. In the following self-coupling is neglected, i.e. $a_{mm} = 0$.

B. Spike-Timing-Dependent Plasticity

In order to include learning, we combine the Lighthouse model with the mechanism of spike time dependent plasticity. For a historical review we refer the reader to [21]. This mechanism is based on the Hebbian learning rule. We will rely on the formulations of C. Chen and D. Jasnaw [7] and van Rossum et al. [22]. The underlying mechanism of STDP is as follows: The coupling weight a_{km} will be strengthened due to causal firing and weakened in the case of acausal firing. Causal firing means that neuron m spikes in advance of neuron k . Acausal firing denotes the reverse case. The reverse coupling weight a_{mk} will be weakened if a_{km} is strengthened and vice versa. Hence, the connections between two neurons become directed and dynamical.

In order to formulate a mathematical model of spike time dependent plasticity one has to design a mechanism, which is able to distinguish causal from acausal spiking. Spiking events of neuron i are encoded in the spike train $S_i = \sum_n \delta(t - t_i^n)$. To the n -th firing event of neuron j at time t_j^n we connect a function

$\sigma_j(t) = \sigma_0 \Theta(t - t_j) e^{-\frac{(t - t_j^n)}{\tau_\sigma}}$. The product $\sigma_j(t) S_i(t)$ is zero if $t_i^n < t_j^n$, whereas it is nonzero in the reverse case. We have to assume that the decay time τ_σ is smaller than the characteristic

inter-spike intervals $t_{j+1} - t_j$. The coupling weight a_{ij} decreases in the first (acausal) case. A similar reasoning can be used for the reversed case. It is convenient to introduce two functions $\sigma_j(t)$, denoted as $A_j(t)$ and $B_j(t)$, for each neuron j .

The variable $A_m(t)$ associated to neuron m is related to strengthening of the connection a_{km} if the firing with respect to neuron k is causal, i.e. if the product $A_m(t) S_k(t)$ is different from zero. The variable $B_k(t)$ induces weakening of the coupling strength a_{km} if the firing with respect to neuron m is acausal, i.e. if the product $B_k(t) S_m(t)$ is different from zero. The dynamics of the coupling weight is then given by the evolution equation

$$\dot{a}_{km} = \Delta A_m S_k - r a_{km} B_k S_m \quad (5)$$

The first term of equation (5) is related to strengthening due to causal firing and the amount is proportional to the potentiation constant Δ . The second term causes depression of the coupling weight. It is proportional to the depression constant r and to the coupling weight a_{km} itself.

In order to determine the functions $A_m(t)$ and $B_m(t)$ dynamically we closely follow the work of Chen and Jasnaw [7]. They used the following evolution equations, where $\sigma_m(t)$ now denotes A_m and B_m . The quantities A_m and B_m can be viewed as concentrations of chemical species, which are generated by a spiking event of the neuron m .

$$\dot{\sigma}_m = u_\sigma (1 - \sigma_m - I_\sigma) S_m - \frac{\sigma_m}{\tau_\sigma} \quad ; \quad \sigma = A, B \quad (6)$$

Furthermore, the dynamics of I_σ is given by

$$\dot{I}_\sigma = \frac{\sigma}{\tau_{l\sigma}} - \frac{I_\sigma}{\tau_{R\sigma}} \quad (7)$$

The spiking of a neuron, encoded in S_m , leads to an increase of the concentrations A_m, B_m . Furthermore, the concentrations decay with time constants τ_σ . For details we refer the reader to [7].

III. STATES OF ENHANCED ACTIVITY

The aim of this article is to find states in the extended Lighthouse model, which exhibit features of epileptic seizures. There seems to be no commonly accepted definition of a seizure in literature. However, as a main feature one may consider the fact that all neurons in a region with epileptic spiking are active and spike with a high frequency. Synchronization in the sense of dynamical systems theory seems to be an accompanying phenomenon, as pointed out by R. S. Fisher et al [23]. However, whether synchronization is the origin of a seizure or plays a major role in the termination, as emphasized by [4, 24], is yet unclear.

Therefore, in our analysis of the generalized Lighthouse model, we use the notion of *states of enhanced activity*, which could be interpreted as an epileptic seizure. A state of enhanced activity is defined by a strong mean dendritic current

$$\Psi(t) = \frac{1}{N} \sum_i \psi_i(t) \quad (8)$$

A plot of the time signal of this mean current is exhibited in fig (2). It is evident that periods of strong mean current and weak mean current interchange. In order to explicitly determine

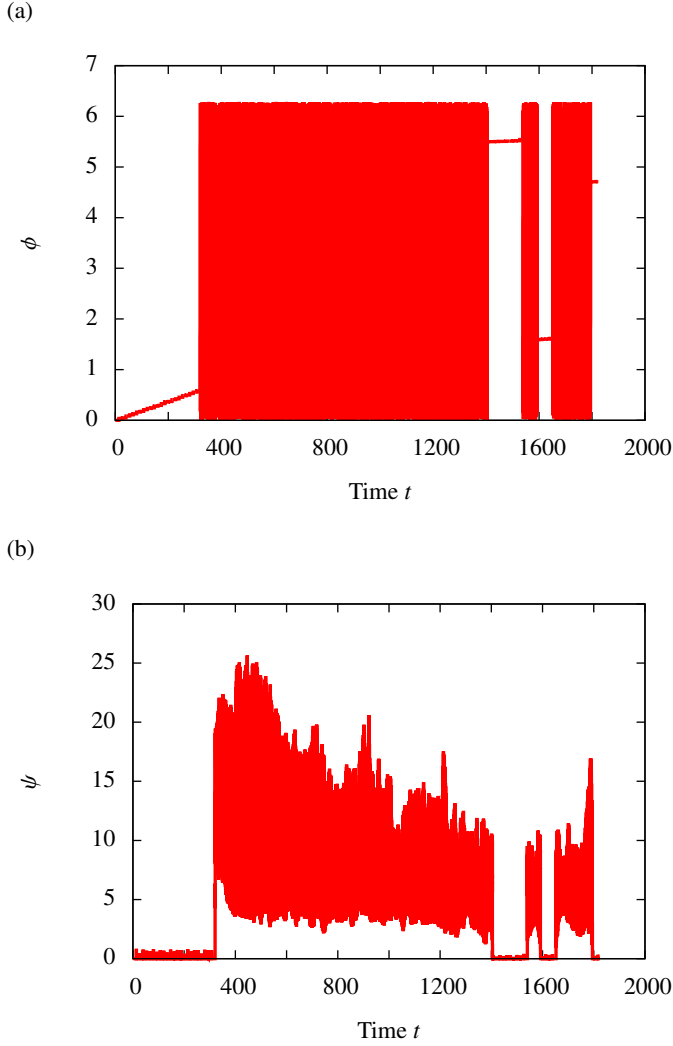


Figure 1: (a) Phase ϕ of a single neuron is shown as a function of time. (b) The dendritic current ψ of the same neuron is plotted in time. Figures (a) and (b) demonstrate the existence of time-intervals, during which the neuron is at rest and intervals when it is highly active.

a state of enhanced activity we introduce a threshold value for the mean current. A closer inspection of the time signals demonstrates that during the state of enhanced activity the spiking frequency of most of the neurons are in the saturated regime, i.e. almost all neurons fire with the highest attainable frequency ν . This is demonstrated in fig (2), in which the mean spiking frequency $\langle \dot{\phi} \rangle$

$$\langle \dot{\phi}(t) \rangle = \frac{1}{N} \sum_i \dot{\phi}_i(t) \quad (9)$$

of the system is shown.

A. Initiation and Termination of States of Enhanced Activity

States of enhanced activity emerge in the neuronal generalized Lighthouse model in the following situations: The coupling weights are initialized randomly which implies that all to all coupling exists. One special neuron is singled out receiving an

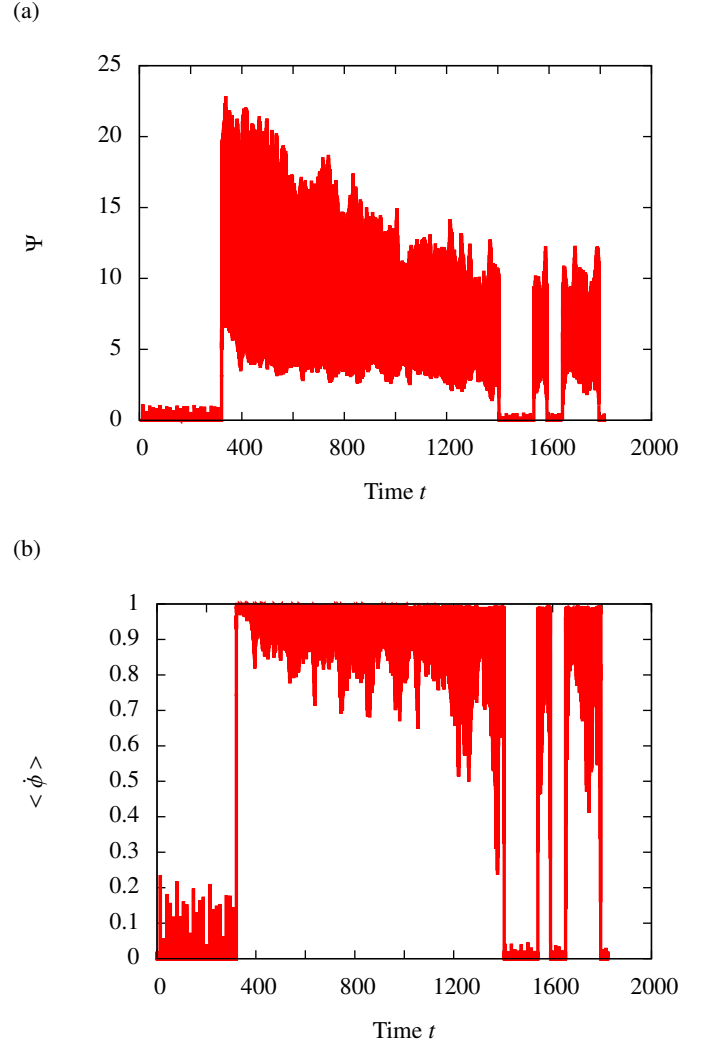


Figure 2: (a) Mean dendritic current $\Psi(t)$ as a function of time. (b) Mean spiking frequency $\langle \dot{\phi} \rangle$ as a function of time.

external signal $p_{(ext,m)}$ consisting of a sequence of delta peaks:

$$p_{(ext,m)} = \sum_n A_{p_{ext}} \delta(t - n\Delta T) \quad (10)$$

The amplitude $A_{p_{ext}}$ of the peaks is chosen in such a way that the externally excited neuron always gains the maximal spiking frequency ν . The period ΔT is chosen to be $2\pi/\nu$.

The external signal mimics an external stimulation of the network. It is well-known that epileptic seizures can be initiated by periodic external stimuli like light flashes.

As one can see in figure (1), time-intervals occur where all neurons emit nonvanishing dendritic currents connected with a rapidly changing phase implying a strong spiking behaviour of the neurons. These time intervals of strong spiking activity are interrupted by intervals where no neuron, except the externally driven one, is active.

States of enhanced activity are initiated by the externally driven neuron. Due to the external forcing this neuron spikes periodically. As a consequence, all neurons receive pulses, whose strength is proportional to the coupling weight. The phases of the neurons pile up until the phase of one of the neurons reaches 2π . Then, this neuron firing leads to an increase of the phase of the other connected neurons. If their phases are close to 2π , they eventually will spike and a chain reaction is initiated, which results in a state of enhanced activity.

If the amplitude $A_{p_{ext}}$ is lowered, the seizure onset time increases. Below a certain threshold, states of enhanced activity are not observed.

Termination of a states of enhanced activity is clearly due to a change of the coupling weights a_{km} . During states of enhanced activity the causal nature of the firing of neurons, which is responsible for the built up of a network structure with strong couplings between causally firing neurons, change to acausal firing. This eventually leads to a weakening of the coupling weights and, hence, to the destruction of the state of enhanced activity.

Let us exemplify this mechanism considering two neurons i and j in more details. Assume that neuron j fires before neuron i . As a result the coupling weight a_{ij} will increase, whereas a_{ji} decreases. Hence, we investigate the following dynamics:

$$\begin{aligned}\dot{\phi}_i &= \Xi(\psi_i, \Theta) \\ \dot{\psi}_i &= -\gamma\psi_i + a_{ij}S_j(\phi_j) \\ \dot{\phi}_j &= \Xi(\psi_j, \Theta) \\ \dot{\psi}_j &= -\gamma\psi_j\end{aligned}\quad (11)$$

We have neglected the coupling $a_{ji} \approx 0$, taking into account that the neuron j spikes before neuron i .

The solution for the dendritic current $\psi_i(t)$ is given in terms of the spike-times T_j^n of neuron j , which are determined by the spiking condition $\Theta_j(T_j^n) = 2\pi$:

$$\psi_i(t) = \sum_n e^{-\gamma(t-T_j^n)} a_{ij}(T_j^n) \quad (12)$$

If we consider a step function $\Xi(\psi, \Theta)$, which is zero if $\psi \leq \Theta$ and ν , if $\psi > \Theta$, the phase $\phi_i(t)$ changes only, if $\psi_i > \Theta$. This is the case if $a_{ij}(T_j^n) > \Theta$.

Due to the acausal situation of neuron j , i.e. due to $a_{ji} \approx 0$ the dendritic current of neuron j decays exponentially, and the spiking frequency decreases

$$\begin{aligned}\phi_j &= \phi_j(0) + \int_0^t dt' \Xi(\psi_j(t'), \Theta) \\ &= \phi_j(0) + \nu \ln \frac{1 + (\frac{\psi_j(0)}{A})^M}{1 + (\frac{\psi_j(0)}{A})^M e^{-M\gamma t}}\end{aligned}\quad (13)$$

Eventually, this leads to a stationary phase $\phi_j(0) + \nu \ln \frac{1 + (\frac{\psi_j(0)}{A})^M}{1 + (\frac{\psi_j(0)}{A})^M e^{-M\gamma t}}$. However, for small values of the damping constant γ , the dendritic current of neuron i remains at a finite value, and, in turn its spiking frequency remains finite, provided the condition $\sum_n e^{-\gamma(t-T_j^n)} a_{ij}(T_j^n) > \Theta$ is fulfilled. As a consequence, the phase Φ_i increases more rapidly than the phase of neuron j : The phase of neuron i overtakes the phase of neuron j rendering the connection a_{ij} acausal. This transition from causal to acausal firing leads to a decrease of the coupling weight a_{ij} and an increase of the coupling a_{ji} .

This observation can be considered to be consistent with the experimental findings of Schindler et al. [24] and Lehnertz et al. [4]. These authors state that for a certain kind of epileptic seizures synchronization and instability occur simultaneously. They argue that the synchronization towards the end of the seizure may be even a self-regulatory mechanism to catalyse the seizure termination.

The described behaviour exists for a certain range of values of the damping constant γ , $\gamma_c^l < \gamma < \gamma_c^u$. This has been investigated

Numerical parameters:	Naka-Rushton relation:
Time step $dt = 0.01$	maximal spiking rate ν : 1.0
	Threshold θ : 10.0
	Steepness M : 3
External signal:	Neuronal network:
Amplitude $A_{p_{ext}}$: 10.0	Number Neurons N : 50
Forcing Periodic $T_{p_{ext}}$: 1.0	Damping constant γ : 0.7
	Enhancement constant c_m : 5.0
Learning parameters:	
Potential constant Δ : 1.0	Depression constant r : 1.0
Potential width τ_A : 0.2	Depression width τ_B : 0.2
Recovery rate $\tau_{r\sigma}$: 10.0	Fatigue rate $\tau_{l\sigma}$: 10.0
Chemical fraction u_A : 0.9	Chemical fraction u_B : 0.9

Table I: Numerical coefficients used in the simulations, are shown.

by a numerical investigation of the generalized Lighthouse model. This range depends on the system size.

The described mechanism only works, if the damping constant γ of the dendritic current is large enough. Therefore a lower critical damping constant γ_c^l exists. On the opposite, the chain reaction can not start, if the damping constant exceeds a certain critical upper value γ_c^u . The neurons will start to spike, but the dendritic current of the neuron is decaying too fast and therefore the phase velocity is not high enough to preserve the firing and initialize a state of enhanced activity.

B. Analysis of Statistical Behaviour

In the following, we present results of the investigation of the statistical behaviour of the states of enhanced activity. In order to create sufficient statistics long time simulations of the generalized Lighthouse model and measurements of the mean dendritic currents have been performed. The used numerical parameters are shown in table (I). 27 runs have been realized, each with $47 \cdot 10^7$ timesteps. In total 10,000 events, which could be classified as state of enhanced activity, have been measured. The duration of a state of enhanced activity, the interevent waiting times between two subsequent states of enhanced activity, and the total energy of a state of enhanced activity have been calculated. The energy of a state of enhanced activity is defined to be proportional to the sum of the squares of all dendritic currents:

$$E = R \left[\frac{1}{N} \sum_i \psi_i(t)^2 \right] \quad (14)$$

Based on this data, we have calculated the probability density functions (PDF) of the energy, the event duration and the interevent waiting time. Furthermore the Omori- and inverse-Omori-Law was determined in the same way as described by Osorio et al. [5]. Furthermore, the conditional expected waiting-time was calculated.

In order to assess the scaling behaviour of the various quantities like energy of an state of enhanced activity, duration of an state of enhanced activity, or the interevent times between two subsequent states of enhanced activity, the scaling coefficients

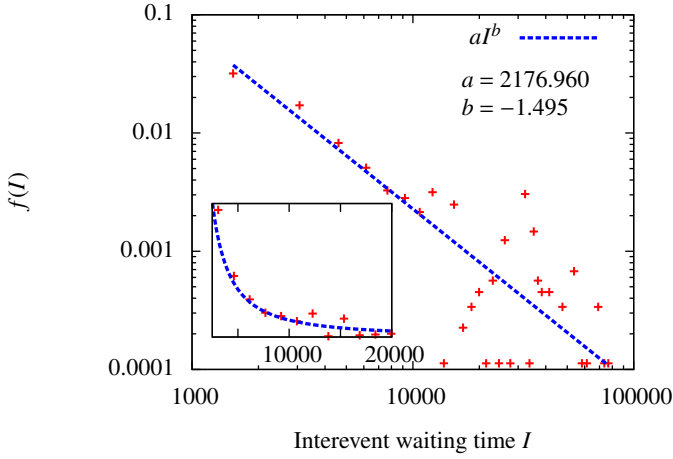


Figure 3: The logarithm of the probability density function $f(I)$ of the interevent-intervals of the states of enhanced activity as a function of $\ln I$. The inset shows the same data points in $f(I)$ - I coordinates. A power law is fitted to the data.

of the various quantities are calculated by the method introduced by Newman [25]. It is based on a maximum likelihood estimation of the scaling coefficient. The estimation is only applied in intervals $[x_{min}, x_{max}]$ of the data in which the power law behaviour holds.

C. Interevent Waiting Time

As stated by Osorio et al and Saichev et al [5, 26], the probability density function of the interevent waiting time for earthquakes is described by a power law with a scaling coefficient of -1.1 . Osorio et al [5] calculated the PDF for data sets of epileptic seizures. They obtained an approximate value of $-\frac{3}{2}$ for the scaling coefficient.

From our numerical computations of the generalized Lighthouse model we extract a scaling coefficient of -1.495 ± 0.149 for $[x_{min} = 100, x_{max} = 18000]$, which perfectly agrees with the analysis of Osorio [5]. The data is exhibited in figure (3). It is obvious that the power law fits quite well the measured behaviour for small and medium interevent waiting times. For larger values of the interevent times the data points are scattered around the fitted power law. This could be explained, following Osorio et al [5], by the assumption that the probability density function exhibits different power law regimes, a possibility which has first been pointed out by [26]. However, it could also be attributed to the restricted sample size. We remind the reader that the behaviour in the regime of large interevent times is of considerable interest with respect to the analysis of extreme events in complex systems.

D. Gutenberg-Richter-Law

The Gutenberg-Richter-law states that the probability density function for the earthquake seismic moment S exhibits a power law distribution. Similarly, the analysis for epileptic seizures by [5] yields a value close to $-5/3$. Furthermore they showed that a power law with the same scaling exponent is obtained for the energy distribution of epileptic seizures. The results of our calculations, which are summarized in figure (4), support

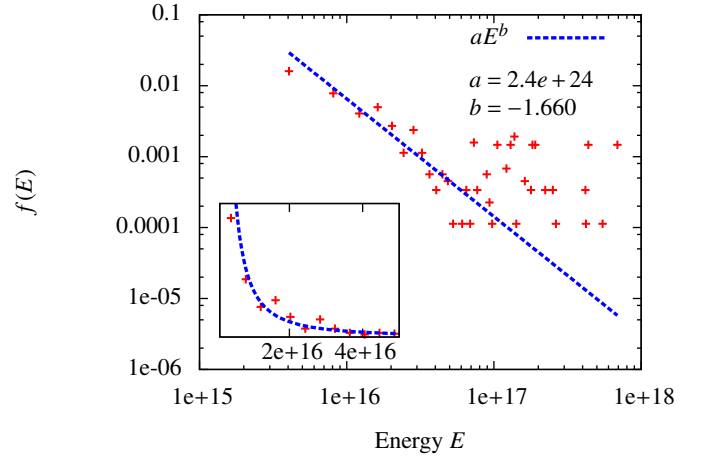


Figure 4: The probability density function $f(E)$ of the total energy of state of enhanced activity is shown. The coefficient is -1.660 ± 0.220 in the interval $[3.7 \cdot 10^{15}, 4 \cdot 10^{16}]$.

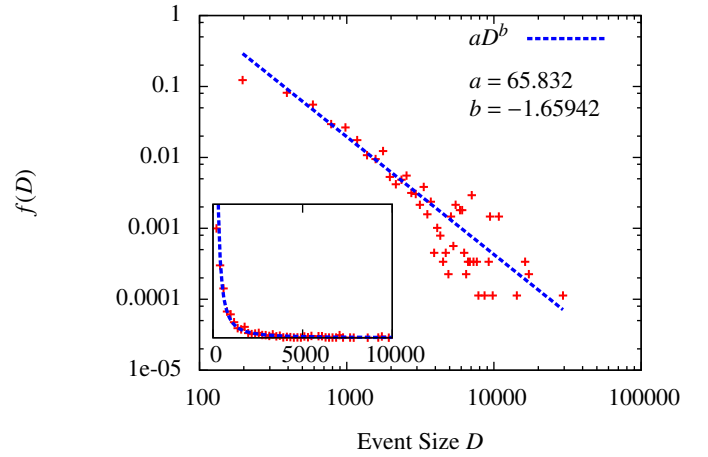


Figure 5: The logarithm of the probability density function $f(D)$ of the duration as a function of $\log D$. The power law fit yields a value for the scaling coefficient of -1.659 ± 0.155 in the interval $[200, 3783]$. The inset exhibits $f(D)$ as a function of D .

this assumption.

As for the statistics of the interevent times the behaviour of the PDF deviates from the powerlaw for high values of E , which can be explained by the same reasonings as discussed above. If one considers the PDF of the duration of the states of enhanced activity, which is plotted in figure (5), one can see that both PDFs have a similar scaling exponent.

Therefore, a direct relationship between energy E and duration of the state of enhanced activity should exist. This relationship can be seen on the basis of the definition

$$E(S) = \frac{R}{M} \sum_{t_a, t_e} \psi_m(t_n)^2 = \langle I \rangle \cdot D(S). \quad (15)$$

of the energy contained in the state of enhanced activity between the time instants t_a, t_e . If we replace approximately the quantity $\psi_m(t_n)^2$ by a mean intensity $\langle I \rangle$ we obtain a direct relationship between E and duration D , and, hence, the same scaling behaviour.

Assuming the validity of this relation, one can conclude that all states, independent of their duration, have the same

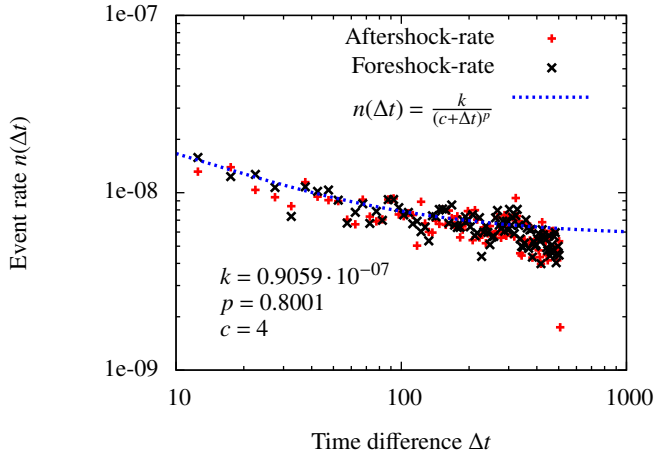


Figure 6: The foreshock and aftershock rates for states of enhanced activity are shown in a double logarithmic scale. The time axis for the foreshock rate is inverted. The Omori-Law is fitted into the data points and the fitting-parameters are shown.

static distribution of the intensity $\langle I \rangle$ and therefore the same distribution of the dendritic current Ψ .

E. Omori Law and Inverse Omori Law

Earthquakes are rare events, which are accompanied by the occurrence of aftershock and foreshock events. The rate $n(\Delta t)$ of these events follows the so-called Omori- and inverse Omori-Law as a function of the time distance Δt from the main event:

$$\log(n(\Delta t)) = \log(k) - p \log(c + \Delta t) \quad (16)$$

Both laws are empirical. These laws originally were introduced by Omori et al [27] and expanded by Utsu et al [28].

Osorio et al [5] pointed out that the Omori- and inverse Omori-Law can also be formulated for epileptic seizures. The analysis of data generated by the generalized Lighthouse model supports the validity of these laws. Our results are presented in figure (6). The Omori-Law is fitted to the data. Both, the foreshock and aftershock rates can be represented by the same fit. This implies that the shock rates are not only described by the Omori-Law, but are symmetric. The results are consistent with the results for data of epileptic seizures [5] and are similar to the earthquake data [5, 29–31].

F. Expected Waiting Time

An interesting question is how the waiting time, which is the time to the next event, depends on the time that has already passed since the last event. The results of our numerical simulations are shown in figure (7).

As one can see, the expected waiting time to the next event increases as a function of the distance to the last event to a maximum. Then the expected waiting time rapidly drops down to zero. The intersection with the x-axis marks the maximal time that can pass before the next state occurs. This is clearly a

finite size effect and depends on the number of neurons of the model. A similar behavior is observed for epileptic seizures as well as earthquakes [5].

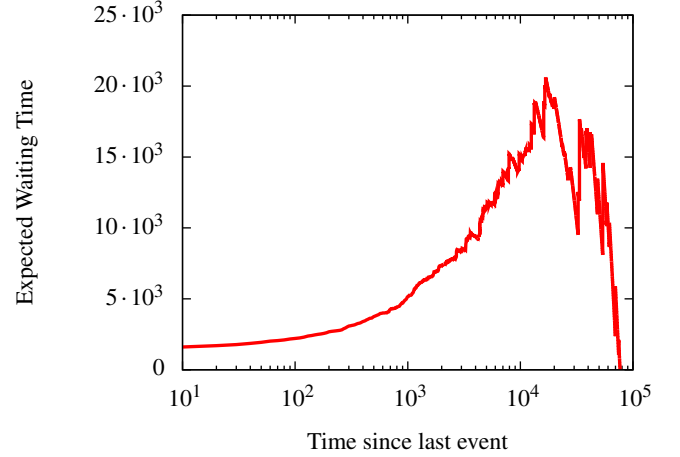


Figure 7: Averaged waiting time to the next seizure-like-state as a function of the time distance to the last event.

IV. SUMMARY AND OUTLOOK

We combined the Lighthouse model introduced by Haken [6] with a STDP-learning algorithm to a model in which states of enhanced activity emerge. It has been shown that these states share characteristic features reported for time signals of epileptic seizures. Defining states of enhanced activity via the energy E we demonstrated the validity of the Gutenberg-Richter law, the Omori and inverse Omori laws and showed similarities in the statistics of inter-seizure intervals. We want to emphasize that our model yields scaling exponents rather close to the ones reported in the work of Osorio et al. [5]. Therefore, a detailed investigation of the generalized Lighthouse model should allow for an analytical assessment of these scaling exponents. Furthermore, we have described the mechanism which leads to a termination of the states of enhanced activity as a transition from causal to acausal firing, connected with an immediate restructuring of the network topology due to STDP. Due to the closeness to experimental findings it would be interesting to explore the phase diagram of this generalized Lighthouse model in more details and analyze experimental data, whether the mechanism of termination of state of enhanced activity can also be detected for epileptic seizures. For the future, we plan to perform an extended analysis in order to assess the transitions and bifurcation scenarios leading to the regime, where state of enhanced activity arise, from ordered or disordered states adding dynamical details to the schematic phase diagram given in Osorio [5] locating seizure states in a control parameter space spanned by the variables termed *interaction strength* and *heterogeneity*. Additionally, we plan to extend our analysis to larger data samples extending our numerical calculations to longer time periods. This will allow us to resolve the PDF's of extreme events, along the lines suggested by Sornette [32]. In this way we want to contribute to the recent discussions on the existence of dragon kings [32, 33] and the possibility of predictions of extreme events, recently reviewed in [34].

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